Regulation of respiration: Chemical regulation

INTENDED LEARNING OBJECTIVES (ILOs)

By the end of this lecture the student will be able to:

1. Identify the location and function of central chemoreceptors and their role in regulation of ventilation.
2. Identify the location and function of peripheral chemoreceptors and their role in regulation of ventilation.
3. Explain the ventilatory responses to increased PCO_2.
4. Describe the ventilatory responses to decreased PO_2.
5. Explain causes of hyperventilation during moderate muscular exercise.

CHEMICAL REGULATION OF RESPIRATION

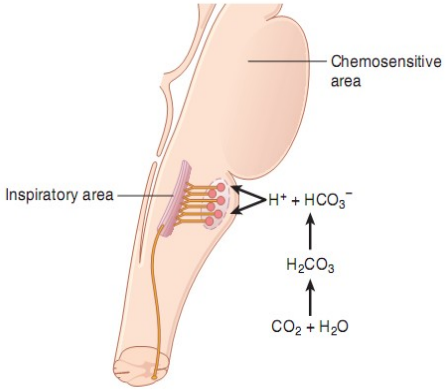
Chemical regulation of ventilation is done by changes in level of CO_2, O_2 and H^+

These changes are mediate by 2 types of chemoreceptors

A- Central:

Located in medulla and stimulated by rise in arterial blood CO_2 which pass through BBB leading to rise CSF H^+, stimulating the central chemoreceptors.

Change in arterial H^+ does not affect central chemoreceptors can't pass BBB.



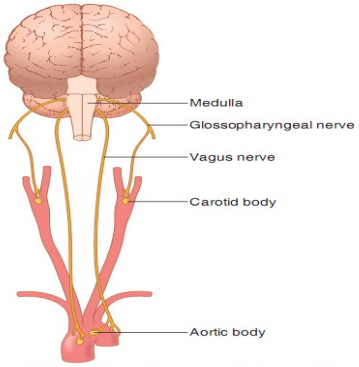


Figure 42-4. Respiratory control by peripheral chemoreceptors in the carotid and aortic bodies.

B- Peripheral:

Located in the carotid and aortic bodies, stimulated by decrease in PO_2 and increase in PCO_2 and H^+ in arterial blood.

N.B: peripheral chemoreceptors respond to decrease in PO_2 and not the O_2 content. So, in anemia and CO poisoning the peripheral chemoreceptors are not stimulated

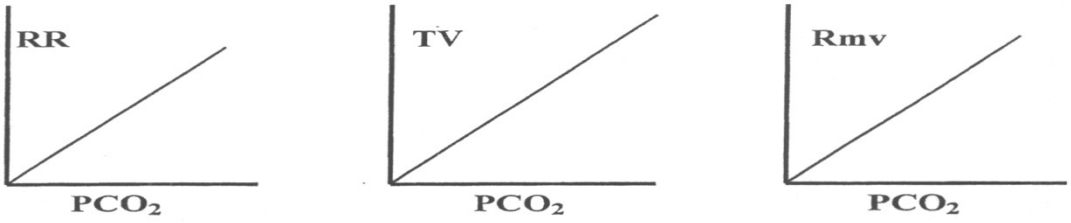
VENTILATORY RESPONSES TO INCREASED PCO_2

Under normal condition arterial PCO_2 is the main regulator of respiration. It contribute to *minute to minute control* of respiration

Change in arterial PCO2 stimulate ventilation reflexly *mainly* through the central *chemoreceptors* and to lesser extend through carotid and aortic bodies which are only weakly responsive to changes in arterial PCO_2

N.B.: ***Increase in PCO_2 beyond*** *70 mmHg*, does not increase ventilation, but actually depress the entire brain *(CO_2 narcosis).*

Response to CO_2:



N.B: ventilation stimulated via peripheral chemoreceptors by a decrease in PO_2 only when the decrease is large.

VENTILATORY RESPONSES TO DECREASED PO_2

Arterial PO_2 is monitored by peripheral chemoreceptors. Arterial PO_2 must ***fall below 60 mm Hg*** before the peripheral chemoreceptors respond by sending afferent impulses to the medullary inspiratory neurons, thereby reflexly increasing ventilation.

It does not play a role in the normal ongoing regulation of respiration, but it is a life-saver mechanism.

N.B: *Decreased arterial PO_2 from 100-60 mm Hg* stimulates peripheral chemoreceptors less than expected, because hyperventilation will decrease both blood PCO_2 and H^+ leading to depression of respiratory centers.

Therefore, the stimulatory effects of hypoxia on ventilation are not clearly manifest until they become strong enough to override the counterbalancing inhibitory effects of a decline in arterial H^+ concentration and PCO_2.

VENTILATORY RESPONSES TO CHANGE IN pH

Change in arterial H^+ concentration can't affect central chemoreceptors, it produces its effect through peripheral chemoreceptors.

Increase in arterial H^+ concentration will lead to hyperventilation and vice versa

CHANGE IN VENTILATION WITH MUSCULAR EXERCISE

1- There is abrupt increase in ventilation with onset of the exercise which may be due to

- Neurogenic signals transmitted directly to respiratory center at the same time that signals go to the muscles to cause contraction.
- Afferent impulses from proprioceptors in muscles, tendons, and joints.

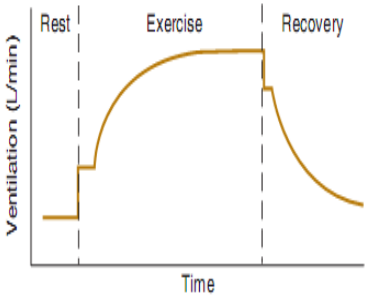


FIGURE 36–14 Diagrammatic representation of changes in ventilation during exercise. See text for details.

2- The more gradual increase is presumably, even though arterial pH, Pco_2, and Po_2 remain constant during moderate exercise and it could be due to:

- The increase in body temperature.
- Exercise increases the plasma K^+ level, and this increase may stimulate the peripheral chemoreceptors.
- The sensitivity of the neurons controlling the response to CO_2 is increased

N.B. In heavy exercise: arterial PCO_2 and H^+ concentration rises at the same time that PO_2 fall, which gives a strong ventilation drive.

SUGGESTED TEXTBOOKS

1. Guyton and Hall textbook of medical physiology, thirteenth edition 2016, Elsevier, chapter 42 , from page 539 to 548
2. Ganong's Review of Medical Physiology, twenty-fifth edition 2016, McGraw-Hill Education, chapter 36, from page 655 to 664
3. Lauralee Sherwood Human Physiology: From Cells to Systems, Ninth edition 2016. CENGAGE, chapter 13, from page 479 to 487